Case Reports

'Yewdow'-Induced Anemia

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GLUCOSE-6-PHOSPHATE DEHYDROGENASE (G6PD) deficiency is a common cause of a sudden unexpected anemic crisis. It is often difficult to trace the exact inciting agent for the hemolytic episode. We report a case of life-threatening hemolytic anemia in a healthy Chinese-Burmese boy who had never been ill to underscore the problem posed by this clinical conundrum.

Report of a Case

The patient, a 2½-year-old boy, had a sudden onset of dark red urine, pallor, and decreased appetite after spending the night with his grandparents. The child had had no previous upper respiratory tract infections, fever, diarrhea, epistaxis, or jaundice. His parents said he had not had exposure to pesticides, prescribed drugs, ethnic herbal medications, teas, or fava beans. The neonatal history was unremarkable for any jaundice, phototherapy, or sudden pallor. Both parents worked in a sewing factory and did not bring their work home.

The child was well developed and pale, with a blood pressure of 107/47 mm of mercury, a pulse rate of 104 beats per minute, and tachypnea with respirations of 40 per minute. Pertinent findings included an intermittent grade I/VI systolic murmur at the left sternal border, poor capillary filling in all extremities, and no organomegaly. He had no petechiae, ecchymoses, or rashes. The hemoglobin concentration was 2.54 mmol per liter (4.1 grams per dl), hematocrit was 0.11 (11%), and the mean corpuscular volume was 102 fl. The uncorrected reticulocyte count (brilliant cresyl blue wet mount) was 68×10^{-3} (6.8%; normal, 1 to 24×10^{-3}), and numerous Heinz bodies were found. A peripheral blood smear revealed moderate anisocytosis and poikilocytosis with fragmented erythrocytes seen. There was pronounced hemoglobinuria, proteinuria, and bilirubinemia. The blood urea nitrogen level was 8.9 mmol per liter of urea (25 mg per dl; normal, 3.0 to 6.5), the serum creatinine was 90 μmol per liter (1.0 mg per dl; normal, 50 to 110), total serum bilirubin was 112 μmol per liter (6.6 mg per dl; normal, 2 to 18) with 5 μmol per liter (0.3 mg per dl) direct-acting bilirubin, alanine aminotransferase was 23 U per liter, aspartate aminotransferase was 98 U per liter, and lactic dehydrogenase was 1,479 U per liter. Serum tests for antibodies to hepatitis A and B, heterophil agglutination, and Coombs' testing were negative. Emergency therapy consisted of transfusion with packed red cells and mannitol- and furosemide-induced diuresis. The patient recovered completely in four days, with a return to normal of his blood chemistry values.

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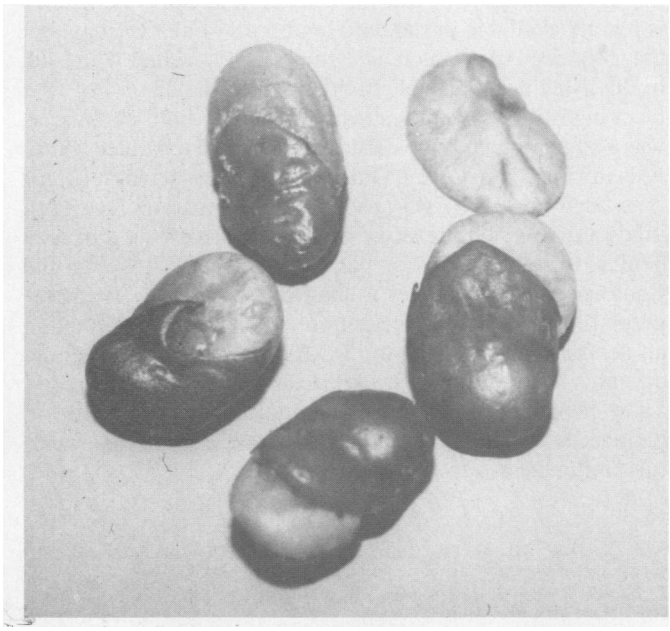

Figure 1.—Several fried fava beans (*Vicia faba* Leguminosae; "yewdow") are shown.

Several months later, the patient was shown to have 0 IU of G6PD enzyme activity per gram of hemoglobin (normal range, 7.9 to 16.3 IU per gram). His hemoglobin at that time was 7.57 mmol per liter (12.2 grams per dl), and his reticulocyte count was 24×10^{-3} (2.4%). Both parents had some G6PD activity detected on a spot test. The parents were again questioned (by a Chinese physician [W.Y.W.]) as to possible ingestions and exposures. The physician asked about "yewdow" ingestion and learned that the child had eaten a packet of this common Asian snack food while watching television with his grandfather. "Yewdow nuts" are prepared from the seed pods of the *Vicia faba* Leguminosae plant, which is the identical species of Mediterranean fava (broad) beans.

Discussion

Glucose-6-phosphate dehydrogenase deficiency is found in persons throughout the world, but is more frequently reported from the tropics and Mediterranean areas. There are more than 150 variants of the G6PD enzyme. The A– variant is found more commonly in blacks. G6PD B– and G6PD Canton are found in populations of the Mediterranean and Southeast Asian regions. Both increased spectrin and decreased glomerular-stimulating hormone levels have been implicated in the mechanism of erythrocyte damage.[1] The selective advantage of G6PD deficiency in malaria-endemic areas was suggested by Allison[2] and Motulsky[3] in the 1960s and further substantiated by Usanga and Luzzatto[4] in the 1980s. This enzyme deficiency has been found to confer an additional antimalarial advantage,[5] but this advantage disappears when a patient is transplanted to an area nonendemic for malaria. Panich has determined the frequency of G6PD deficiency in several Asiatic populations.[6] Of particular importance to physicians in California is the fact that 5% of Southeast Asian male refugees to the United States had decreased G6PD activity.[7]

Incriminated agents inducing hemolysis include the well-known sulfonamides and antimalarial drugs, nitrofurantoin, infections such as hepatitis,[8] and unripe peaches.[9] An extensive list of compounds causing hemolysis in G6PD deficiency is readily available in standard textbooks of hematology, but the frequency of appreciable hemolysis associated with each of the listed agents is not. In this patient, the history of fava bean ingestion was emphatically denied by both parents, as was a large number of possible agents known to induce hemolysis in G6PD deficiency. Only when the vernacular term for fava bean (yewdow) was used was a history of fava bean ingestion obtained. Yewdow is a popular snack food in Asia similar to potato chips or peanuts in America. Within the Cantonese community, it is known as *chundow.* A photograph of the beans may be helpful when attempting to elicit an accurate history (Figure 1). As the cultural and ethnic diversity increases in the western United States, added knowledge of these differences is required to properly inform patients of pertinent inciting agents to avoid within their cultural milieu.

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Neurogenic Diabetes Insipidus in an Initially Healthy Neonate

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NEUROGENIC DIABETES INSIPIDUS in the newborn period is rare but has been described previously in neonates with infections, congenital brain malformations, intraventricular hemorrhage, and hypoxia.[1-4] This report concerns a 21-day-old infant with central diabetes insipidus in whom no predisposing factors were evident at the time of diagnosis.

Report of a Case

The patient, a 21-day-old female infant, was born to a 20-year-old primigravid woman at 38 weeks' gestation after an uneventful pregnancy. The vaginal delivery was without complication. The infant weighed 3,200 grams (7 lb 1 oz) at birth. She had mild transient tachypnea of the newborn that did not require supplemental oxygen and resolved spontaneously within 12 hours. The baby nursed well while in the hospital and was discharged home on the third postpartum day, weighing 3,062 grams (6 lb 12 oz). Office visits at 10 days and 20 days of age dealt with parental concerns of feeding difficulties and hard stools. Because of progressive vomiting, diarrhea, lethargy, and irritability, however, the infant was admitted to hospital at 21 days of age for evaluation. The parents had not noted the baby to have fever. Of significance was that they had been feeding the patient strained water boiled with rice and caraway seeds, which the infant preferred over formula or breast-feeding.

On physical examination, the infant was lethargic. Her temperature and vital signs were normal, and she weighed 3,200 grams (7 lbs 1 oz). The general physical findings were within normal limits, with the exception of dry mucous membranes and decreased skin turgor, indicating an approximately 5% dehydration. The initial laboratory studies elicited the following values: serum sodium 157 mmol per liter, potassium 4.9 mmol per liter, chloride 127 mmol per liter, carbon dioxide 16 mmol per liter, blood urea nitrogen 8 mmol per liter (normal, 3.0 to 6.5), and creatinine 71 μmol per liter (0.8 mg per dl [normal, 50 to 110 μmol per liter]). A leukocyte count was 11.6×10^9 per liter with polymorphonuclear neutrophils 0.22 and lymphocytes 0.72. The hematocrit was 0.48. The initial urinalysis revealed a specific gravity of 1.003, and the results were otherwise normal. A sepsis workup consisting of urine, cerebrospinal fluid, and blood cultures was negative for pathogens.

The infant was hydrated with an intravenous solution of 5% dextrose-isotonic fluid at 150 ml per kg per day after an intravenous bolus of 10 mg per kg of body weight. By 18 hours after admission, the infant's hydration status was slightly improved. Her weight had increased only 50 grams to 3,250 grams, and the urine output remained elevated at 4.3 ml per kg per hour. The child remained somewhat lethargic, and the electrolytes were only modestly corrected, with a serum sodium level of 151 mmol per liter, potassium 4.1 mmol per liter, chloride 129 mmol per liter, and CO_2 22 mmol per liter. Serum and urine osmolalities were 480 and 50 mmol per kg, respectively. Despite intravenous rehydration, her weight 24 hours later had increased only 35 grams and her urine output remained high at 5.6 ml per kg per hour. Diabetes insipidus was suspected, and a diagnostic argipressin (8-α-arginine vasopressin) infusion was administered intravenously at a rate of 0.3 units per kg per hour over a period of ten hours. Twelve hours after the infusion was started, the serum and urine osmolalities were 288 and 485 mmol per kg, respectively, and the urine specific gravity had risen from 1.003 to 1.022. The infant's weight increased 75 grams to 3,335 grams, and the urine output decreased to 3 ml per kg per hour. Treatment with desmopressin acetate, 0.05 ml given intranasally each morning, was begun, resulting in a return to normal of her serum electrolytes and urine specific gravity. The following day her weight had increased another 85 grams to 3,420 grams. Because of an increased nighttime urine output, however, and a nocturnal decrease of the urine specific gravity, the dosage of desmopressin was changed to 0.025 ml twice a day. Further evaluation for the source of the diabetes insipidus was undertaken and revealed the following normal results: a random serum cortisol level

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